CURRENT LISTING OF CLAIMS

The following listing of claims, with markings to show any changes made, will replace all prior versions, and listings, of the claims in the application.

Claims 1-36 (Canceled)

37. (Currently Amended) A method for reducing an ~~antigen-specific~~ epitope-specific immune response against an antigenic determinant in a subject, comprising:

administering to the subject a composition comprising a therapeutically effective amount of a purified MHC Class II polypeptide comprising covalently linked first and second domains, wherein:

the first domain is a human MHC class II β1 domain and the second domain is a mammalian MHC class II α1 domain and wherein the amino terminus of the second domain is covalently linked to the carboxy terminus of the first domain and wherein the MHC class II polypeptide does not include an α2 or β2 domain, wherein the MHC Class II polypeptide is non-covalently associated or covalently conjugated with the antigenic determinant; and

wherein administration of the MHC Class II polypeptide associated or conjugated with the antigenic determinant reduces the ~~antigen-specific~~ epitope-specific immune response against the antigenic determinant in the subject.

38. (Original) The method of claim 37, wherein the reduced immune response is a decrease in an influx or proliferation of a T cell, a macrophage, a B cell, or an NK cell.

39. (Original) The method of claim 37, wherein the reduced immune response is a reduction in the expression of a cytokine.

40. (Original) The method of claim 37, wherein the reduced immune response is an induction of a T suppressor cell response.

Claims 41-53 (Cancelled)

54. (Currently Amended) A method of treating a disease caused by ~~antigen-specific~~ epitope-specific T–cells, comprising;

administering to a patient presenting with said disease or at an elevated risk for developing the disease a composition comprising a therapeutically effective amount of a purified MHC Class II polypeptide comprising covalently linked first and second domains, wherein the first domain is a human MHC class II β1 domain and the second domain is a mammalian MHC class II α1 domain and wherein the amino terminus of the second domain is covalently linked to

the carboxy terminus of the first domain and wherein the MHC class II molecule does not include an $\alpha 2$ or $\beta 2$ domain, and wherein the MHC Class II polypeptide is non-covalently associated or covalently conjugated with an antigenic determinant,

wherein an ~~antigen-specific~~ epitope-specific response by said T-cells directed against said antigenic determinant is a causal or contributing factor in said disease;

whereby said T-cells following exposure to said MHC Class II polypeptide and associated or conjugated antigenic determinant mediate or exhibit a reduction of ~~antigen-specific~~ epitope-specific T-cell pathogenic activity or potential in the patient thereby treating the disease.

Claims 55-58 (Cancelled)

59. (Previously Presented) The method of claim 54, wherein the disease caused by antigen-specific T-cells is rheumatoid arthritis, chronic beryllium disease, insulin-dependent diabetes mellitus, throiditis, inflammatory bowel disease, uveitus, polyarteritis, Multiple Sclerosis or Myasthenia Gravis.

60. (Previously Presented) The method of claim 54, wherein the disease is an autoimmune disorder.

61. (Previously Presented) The method of claim 60, wherein the disease is Multiple Sclerosis.

62. (Previously Presented) The method of claim 54, wherein the covalent linkage between the first and second domains is provided by a peptide linker sequence.

63. (Previously Presented) The method of claim 54, wherein the polypeptide further comprises, covalently linked to the amino terminus of the first domain, a third domain comprising an antigenic determinant.

64. (Previously Presented) The method of claim 63, wherein the antigenic determinant is a peptide antigen.

65. (Previously Presented) The method of claim 63, wherein the covalent linkage between the first and third domains is provided by a peptide linker sequence.

66. (Previously Presented) The method of claim 54, wherein the antigenic determinant is associated with the polypeptide by non-covalent interaction.

67. (Previously Presented) The method of claim 66, wherein the antigenic determinant is a peptide antigen.

68. (Previously Presented) The method of claim 54, wherein the polypeptide further comprises a covalently linked detectable marker or toxic moiety.

69. (Previously Presented) The method of claim 37, wherein the subject has rheumatoid arthritis, chronic beryllium disease, insulin-dependent diabetes mellitus, throiditis, inflammatory bowel disease, uveitus, polyarteritis, Multiple Sclerosis or Myasthenia Gravis.

70. (Previously Presented) The method of claim 37, wherein the subject has an autoimmune disorder.

71. (Previously Presented) The method of claim 37, wherein the subject has Multiple Sclerosis.

72. (Previously Presented) The method of claim 37, wherein the covalent linkage between the first and second domains is provided by a peptide linker sequence.

73. (Previously Presented) The method of claim 37, wherein the antigenic determinant is covalently linked to the amino terminus of the first domain of the MHC Class II polypeptide.

74. (Previously Presented) The method of claim 73, wherein the antigenic determinant is a peptide antigen.

75. (Previously Presented) The method of claim 73, wherein the covalent linkage between the first and third domains is provided by a peptide linker sequence.

76. (Currently Amended) The method of claim 54, wherein the reduced ~~antigen-specific~~ epitope-specific T-cell pathogenic activity or potential in the patient following exposure of said T-cells to said MHC Class II polypeptide and associated or conjugated antigenic determinant includes one or more activity(ies) selected from: i) reduced T-cell sensitivity to pathogenic stimulation by endogenous antigen; ii) T-cell secretion of an anti-inflammatory cytokine; iii) T-cell acquisition or induction of a T-supressor phenotype; iv) reduction or prevention of antigen-stimulated T-cell proliferation; v) reduction or prevention of inflammatory T-cell migration or recruitment to a selected tissue or compartment in the subject; vi) T-cell anergy, and vii) T-cell apoptosis.

77. (Previously Presented) The method of claim 76, wherein the disease is an autoimmune disorder.

78. (Previously Presented) The method of claim 37, wherein the MHC class II polypeptide is covalently linked with a detectable marker or toxic moiety.

79. (Previously Presented) The method of claim 37, wherein the antigenic determinant comprises a myelin basic protein.

80. (Previously Presented) The method of claim 79, wherein the myelin basic protein comprises SEQ ID NO: 25, SEQ ID NO: 26, or SEQ ID NO: 30.

81. (Previously Presented) The method of claim 64, wherein the antigenic determinant comprises a myelin basic protein.

82. (Previously Presented) The method of claim 79, wherein the myelin basic protein comprises SEQ ID NO: 25, SEQ ID NO: 26, or SEQ ID NO: 30.